

**PREVALENCE OF SHOCK AND ITS OUTCOME IN
CHILDREN ADMITTED TO MEDICAL COLLEGE
HOSPITAL, TIRUNEVELI**

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CERTIFICATE

Certified that this dissertation entitled **“PREVALENCE OF PEDIATRIC SHOCK, ETIOLOGICAL CLASSIFICATION, SEVERITY AND OUTCOME OF MANAGEMENT IN CHILDREN ADMITTED TO MEDICAL COLLEGE HOSPITAL, TIRUNEVELI”** is a bonafide work done by **Dr.S.GOBINATHAN M.D** post graduate student of Pediatric Medicine, Tirunelveli Medical College Hospital, Tirunelveli 2008 – 2010.

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INTRODUCTION

Shock¹² is a clinically diagnosed altered physiological status defined as a complex state of circulatory dysfunction that results in inadequate delivery of oxygen and metabolic substrates to the tissues as described by **Adam. J.Schwarz et al in e med.**

Clinical manifestations are due to decreased perfusion to tissues, the compensatory mechanisms that are triggered by the decreased perfusion and the inadequate removal of metabolic wastes.

Shock accounts for 2%¹⁰ of children admitted to Pediatric casualty worldwide as per most western literature and in Nelson text book of Pediatrics. About 10 million children die of shock every year in the world. Highest mortality is observed in under 5 children in developing countries.

Shock may be due to¹¹

- Decreased blood oxygen carrying capacity with acute and profound decrease in hemoglobin, eg. Hemorrhage.
- Unavailability of oxygen eg: Respiratory illness, pulmonary
- Pumping (eg: Myocardial dysfunction) or plumbing failure (eg: distributive, septic shock) failure.

This results in

| | | |
|----------------|----------|----------------------------------|
| Tissue hypoxia | Relative | eg: Septic shock |
| | | O ₂ demand is high |
| | Absolute | eg: Hemorrhagic |
| | | O ₂ available is less |

Anaerobic metabolism

ATP Production + Lactic acid production

H⁺ ion accumulation

+ Accumulated metabolic products

Cellular death / Apoptosis

In order to prevent cellular death, once lactic acidosis sets in, various compensatory mechanisms come into play. The neural and humoral receptors are activated by decreased perfusion and decreased oxygen concentration in the blood and result in an increase in heart rate and stroke volume and help preserve the blood flow to brain, heart and kidneys. Respiratory rate also increases to compensate for metabolic acidosis. Oxygen extraction is increased. All these mechanisms defend the blood pressure and circulation to vital organs. This state of shock is called **Compensated Shock**. **Decompensated Shock** occurs when cardiovascular system fails to maintain the blood pressure in addition to the tissue perfusion. Progression and perpetuation of shock from this state

leads to multi organ failure and death as aptly described by **Ayse Aklan Aritan et al in Signae Vitae**. This irreversible shock¹ as the name implies is the point of no return when mortality rate is high irrespective of interventions. Thus it is necessary to recognise and implement early intervention at the stage of tissue hypoperfusion rather than when hypotension has set in.

Frankel LR Mathers LH. Schok In Nelson Textbook of pediatrics describe multiple end organ dysfunction¹¹ as evidenced by

- Blood pressure below 5th percentile / Requirement of inotropes.
- Platelet count < 80,000 cells /cu.mm
- > 50% Fio₂ requirement to maintain Spo₂
- GCS 11
- Serum creatinine >2 times upper normal limit.
- Serum bilirubin >4 mg%

Transaminases >2 times upper normal limit.

Etiology¹² for shock is varied. Most common cause of shock seen in pediatric patient is hypovolemia. The classification of shock based on the cause is as follows.

1. Hypovolemic - Diarrhea, Vomiting, Hemorrhage, burns
2. Cardiogenic - Congenital valvular heart diseases, cardiomyopathy, myocarditis, scorpion sting, etc.
3. Septic - A combination of hypovolemic, cardiogenic and distributive shock.
4. Distributive - Anaphylactic, Scorpion sting – Neuronal injuries.
5. Obstructive - Tension pneumothorax, cardiac tamponade.
6. Dissociative - Severe anemia, co-poisoning, methemoglobinemia.

On the back ground of clinical presentation, with a high index of suspicion, early shock can be diagnosed using rapid cardiopulmonary assessment⁷ according to the Pediatric Advanced Life Support Guidelines as follows.

- | | | |
|-----------|---|------------------------------------|
| Airway | - | Stable / unstable / obstructed |
| Breathing | - | RR |
| | | Nasal flare / Grunting / Stridor / |
| | | Retractions/ Abdominal / Thoracic |
| | | Air entry |

| | | |
|-------------|---|---|
| | | Creps /wheeze |
| | | Cyanosis |
| Circulation | - | HR |
| | | Pulse volume: difference between central & peripheral pulses |
| | | Warmth / cool below thigh/ knee / ankle |
| | | Capillary refill time |
| | | Blood pressure |
| | | Liver span |
| Disability | - | Alert / Verbal / Pain responsive / |
| | | Unresponsive |
| | | Pupils |
| | | Eye movements & position |
| | | Tone / posture |

This rapid cardiopulmonary assessment provides the best tool for decision making in emergency management. Most effective and sensitive physiologic status monitoring repeatedly by a competent and experienced physician cannot be replaced by the best monitors^{6,7}.

Once diagnosed shock has to be managed aggressively. First hour is considered the golden hour¹. Evaluation and treatment of underlying cause should proceed simultaneously. Airway must be managed as necessary. All children with shock must be administered high flow oxygen as there is tissue hypoxia. Intubation⁸ may be required in the following situations.

- Unstable / obstructed airway
- RR >80/min, Decreased vital capacity saturation <90% with supplemental oxygen.
- Cardiogenic shock
- Decompensated shock
- Septic shock requiring > 40 to 60ml/kg of fluids
- Severe metabolic acidosis
- Low GCS

Vascular access⁸ must be achieved rapidly. If not after 90 seconds, intraosseous route could be used to administer isotonic fluids which are the first choice fluids for correction of shock. Rapid boluses of RL or NS at 20 ml/kg in 5-10min is given. Reassessment is done and further fluids administered depending on the clinical situation. Significant reduction in

mortality is achieved when $>40\text{ml /kg}^4$ of isotonic fluids are administered in the first hour.

No difference in occurrence of ARDS due to rapid fluid bolusing has been noticed in between groups of patients who were given large boluses and groups given lower volumes³.

Protocol for management of shock as given by guidelines of Pediatric Advanced Life Support Guidelines⁸ is as follows.

0min: Assess. Recognise shock in critically ill child

5min: Airway. Stable – 100% O_2 through non rebreathing mask
unstable / Bradypnea – mechanical ventilation with
Bag valve mask.

Consider early intubation.

Circulation: Establish venous access. If difficult intraosseous
access.

Infuse isotonic fluids – 20ml /kg over 15-20 min

Perform rapid cardio pulmonary assessment after each fluid bolus.

Correct documented hypoglycemia and hypocalcemia

Fluid responsive

No improvement

Improved

- Consider further fluid boluses upto 40-200ml/kg in Hypovolemia, sepsis or Anaphylaxis Initiate inotropes and titrate volume. Intubate if required.
- Asthma, status epilepticus, scorpion sting and submersion injuries require only 20-30ml /kg
- In case of bleed, blood transfusion required.
- In DKA with shock restrict bolus to 20 ml/kg to 1-2hrs or 40ml kg/4hr.
- In cardiogenic shock 5-10ml /kg (max 20ml /kg)
- Dopamine at 10mcg/kg/min if blood pressure low
- Dobutamine at 10mcg/kg/min if blood pressure high.
- Epinephrine at 1mcg / kg / min if blood pressure low or following cardiopulmonary arrest.

Maintaining intravascular volume is the key aspect of successful resuscitation¹.

9 fold¹ increase in improvement of survival rate is achieved when early aggressive goal directed resuscitation was initiated.

Isotonic fluids are the first choice fluids for fluid resuscitation of children with shock²². Ringer's lactate or Normal saline are the fluids of choice. In children with diarrheal dehydration and hypovolemic shock, risk of hypernatremic metabolic acidosis exists and here Ringer's Lactate would be more suitable.

In Dengue shock syndrome rushing in²⁸ fluids through leaky capillaries into the interstitial spaces is dangerous and development of and pulmonary edema mitigates against rapid fluid resuscitation.

Colloids are theoretically beneficial but not cost effective, cause coagulation and allergic reactions.²⁶

Whole blood transfusions are also not readily available and fraught with risk of HIV transmission but useful in hemorrhage²⁵.

Dopamine and Dobutamine are the inotropes used in fluid refractory shock due to varied etiology. When low mixed venous oxygen saturation and myocardial dysfunction is suspected Dobutamine is more appropriate for improving cardiac index³¹.

In patients who are resistant to inotropes and fluid resuscitated, Milrinone will improve cardiovascular function along with

catecholamines No adverse effects are observed with this form of therapy^{32,18}.

Earliest sign of reversal of shock is a decline¹ in heart rate. Other therapeutic goals to be achieved are as follows⁶.

CRT < 2 sec

Normal pulses

Warm extremities

Urine output > 1ml/kg/hr

Normal mental status

Decreased lactate

Increased base deficit

Mixed venous saturation > 70%

Though blood pressure falls only late in the course of shock, HR / SBP ratio called the **Shock Index**¹ is useful as an indicator of improving perfusion.

Transfer²¹ to a tertiary care centre is strongly recommended for children in septic shock who require invasive monitoring.

REVIEW OF LITERATURE

A prospective study titled '**A clinical profile of shock in children in Punjab, India**' by **Daljit Singh, Puneet Aulakh Pooni, Atul Chopra and R.C. Bhatia. Jan24, 2006** was conducted at Department of Pediatrics Dayanand Medical College & Hospital, Ludhiana Punjab.

The study was conducted to determine frequency, etiology, type and outcome of shock in children as presenting to a tertiary care referral hospital in Punjab.

Children who had tachycardia and/or hypotension along with signs of systemic hypoperfusion were included into the study. Severity of shock was assessed as compensated or decompensated and etiological classification was also done. Management was initiated on basis of PALS guidelines and Text book of Pediatric Intensive Care, Roger.

Statistical analysis of the data collected was done using Z – test and t-test.

In this study of 98 children who presented with shock, almost 40% were infants. Commonest cause of shock was hypovolemic due to diarrheal dehydration. 60% of the cases had compensated shock. Only 3 children grew organism in blood culture among the septic shock cases. Survival rate was 74% and maximum in hypovolemic shock and least in cardiogenic shock.

In another study conducted by **Chang et al in 1999** to study outcome of shock in 22 pediatric cases. 11 cases were septic shock, 7 hypovolemic and 4 cardiogenic of which 82%, 0% and 75% respectively died.

Surviving sepsis campaign; International guidelines for management of severe sepsis and septic shock:2008 published in Crit. Care Med 2008 Apr;36(4): 1394 – 6 recommends evidence based recommendations regarding the acute management of sepsis and septic shock is the first step towards improved outcomes for this group of critically ill children.

Application of a simple clinical algorithm to guide management could bring about significant improvements in mortality for these patients with shock was aptly evidenced by the **UK audit published in Archives disease of childhood published on 12.01.2009 summarised by Nicola Pocock.**

Carcillo JA, Kuch BA, Han YY, Day S from University of Pittsburgh school of Medicine, Children's Hospital of Pittsburg, Pennsylvania, USA concluded that use of **Pediatrics Advanced Life Support recommended interventions** along with other measures, played important role in reducing mortality and functional morbidity in children being treated for shock by community physicians.

Another prospective study by **Joe Brierley et al published in October 2008 Pep.Vol 122 No:4 P 752-759 of PEDIATRICS** observed that children with Septic shock presented with fluid resistant shock (>40ml /kg)

Journal of intensive care medicine 32 7.7.06 995-1003 identified a significant decline in mortality when >40ml/kg of isotonic fluid was given in the first hour of presentation.

Goh A; Luml in J paediatrics child Health 01 – Oct 1999 35(5): 488 – 92 retriates the distinct risk of mortality and risk of multiple organ dysfunction among critically ill children with shock in their study.

Parkland memorial Hospital and children Medical Center of Dallas study to study the effects of Milrinone in children with septic shock concludes that in all volume resuscitated pediatric patients with septic shock, in addition to catecholamine, Milrinone will improve cardiovascular function.

STUDY JUSTIFICATION

About 10 million children die of shock due to various etiologies each year. Shock is one of the most dramatic, dynamic & life threatening problems in critical care pediatrics. Early recognition and timely intervention are critical for successful treatment of pediatric shock. A strong index of suspicion and rapid cardio pulmonary assessment by the treating physician followed by early aggressive fluid resuscitation could make the difference between life and death for the child who presents with shock.

Such a study published in Pediatrics on call titled outcome of Pediatric shock in Punjab in 2006 by Daljit Singh et. al was conducted. Over all survival was 73.6% and 63% of children with decompensated shock died.

This prospective study would help in identifying the prevalence of shock in a medical college hospital and the outcome of management based on a protocol could be measured. Moreover there is paucity of data on the epidemiology of shock in developing countries⁶.

OBJECTIVE OF THE STUDY

To assess the prevalence of pediatric shock in children admitted to Pediatric ICU, to identify possible etiology and the response to treatment and outcome in patients admitted with shock in Pediatrics department of Tirunelveli Medical College Hospital.

MATERIALS AND METHODS

Study design:

Descriptive study using cross sectional survey methods.

Sample size and sampling techniques:

Sample size : 50

$$q = 1-p$$

$$t = 20\% p$$

Using the formula $4pq/t^2$ to calculate incidence

Study place : Pediatric intensive care unit
Tirunelveli Medical college Hospital,
Tirunelveli.

Inclusion criteria : All patients between ages of 1 month and
12 years admitted to Pediatrics ward,
Tirunelveli Medical college Hospital.

Exclusion criteria : Neonates are excluded from the study

Study period : November 2008 to September 2009

Ethics committee permission at Tirunelveli Medical College was obtained prior to the study.

Study Methodology :

All sick children admitted to Pediatric intensive care unit of Tirunelveli Medical College Hospital with the suspicion of shock are assessed by using the rapid cardiopulmonary assessment and diagnosed as suffering from shock. Possible etiology, type and severity of shock would be arrived at using a targeted history, clinical examination and relevant laboratory investigations.

These children are managed as per the **Pediatric Advanced life support guidelines for shock** with modifications for individual cases as necessary. The outcome of treatment is studied.

Children are classified based on severity as compensated or decompensated shock and based upon their etiology as Hypovolemic, cardiogenic, septic, Distributive, Anaphylactic or Obstructive.

Proforma for dissertation in Pediatric Shock

S.No.:

Date:

Patient Name :

IP No:

Age :

DO Admn.

Sex :

Final diagnosis:

History :

| | | | |
|---------------------------|--|----------------------|--|
| FEVER | | COUGH & COLD | |
| LETHARGY | | RESPIRATORY DISTRESS | |
| COOL EXTREMITIES | | CYANOSIS | |
| DECREASED URINE OUTPUT | | LOOSE STOOLS | |
| ALTERED SENSORIUM | | TRAUMA | |
| SEIZURES | | BLEEDING | |
| POSTURING | | ABSCESSSES | |
| DRUG INTAKE | | SCORPION STING | |
| BEE STING | | SNAKE BITE | |

Past history :

Known heart disease

Chronic /recurrent diarrhea

On Examination:

Airway: Stable/Unstable Maintainable or not

Respiratory rate:

Stridor Grunting SCR/ICR

Air entry Creps/wheeze Color

Heart rate

SIS2 Murmur Cool below

Central peri.pulse difference CRT

BP Liver span Urine output

| | | |
|------|----------|-----------|
| GCS | Pupils | DEM/EOM |
| Tone | Seizures | Posturing |

Treatment details :

| | | |
|-----------------------------|---------|-------------|
| O2 | BVM | Intubations |
| IVF | ml over | |
| IV Dopamine/Dobutamine | | |
| IV Adrenaline/Noradrenaline | | |
| IV Vasodilators | | |

INVESTIGATIONS:

| | | | | |
|---------------|------|---------------|----------|---|
| Sugar | Urea | Creatinine | Na | K |
| Bilirubin | SGOT | SGPT | Proteins | |
| Blood culture | | Urine culture | | |
| CXR | | ECG | Other | |

Course of illness while management

| | | | | | | | |
|------------------|--|--|--|--|--|--|--|
| Time | | | | | | | |
| Airway | | | | | | | |
| RR | | | | | | | |
| Grunting | | | | | | | |
| SCR/ICR | | | | | | | |
| Air entry | | | | | | | |
| Creps/wheez e | | | | | | | |
| Color | | | | | | | |
| SaO2 | | | | | | | |
| HR | | | | | | | |
| Cool below | | | | | | | |
| Central peri | | | | | | | |

| | | | | | | | |
|---------------|--|--|--|--|--|--|--|
| Pulse diffn. | | | | | | | |
| CRT | | | | | | | |
| BP | | | | | | | |
| Liver span | | | | | | | |
| GCS | | | | | | | |
| Pupils | | | | | | | |
| DEM/EOM | | | | | | | |
| Tone | | | | | | | |
| Posturing | | | | | | | |
| Seizures | | | | | | | |
| Urine output | | | | | | | |
| Interventions | | | | | | | |

Outcome: Etiology :

Classification :

MODS :

Outcome of therapy :

Investigator:

Title: _____

IP No: _____

Telephone: _____

Date form completed: _____

OBSERVATIONS

This study aimed at assessing the prevalence of pediatric shock, the etiological profile and the management outcome.

Children diagnosed to have shock by clinical cardiopulmonary assessment were classified according to etiology and severity and managed appropriately as per PALS guidelines and the outcome of management studied.

The data obtained were classified, analyzed and interpreted with the help of statistical package S.P.S. S (13.0) at the 5% level of significance.

Results and Discussion:

I Description of the demographic profile of the study subjects.

The subjects were studied and described according to their demographic characteristics namely sex and age.

The total No. of Pediatric shock cases was 57. Among them 32 (56%) were male and 25 (43.9%) were females.

Table:1

Age and sex wise classification of trials.

| Age Group | Male | | Female | | Total | |
|------------|----------------|--------|----------------|--------|----------------|--------|
| | No | % | No | % | No | % |
| <12 mo | 15 | 46.9 | 12 | 48.0 | 27 | 47.3 |
| 1 – 5 yrs | 5 | 15.6 | 7 | 28.0 | 12 | 21.1 |
| 5 – 10 yrs | 8 | 25.0 | 4 | 16.0 | 12 | 21.1 |
| >10yrs | 4 | 12.5 | 2 | 8.0 | 6 | 10.5 |
| Total | 32 | 100.00 | 25 | 100.00 | 57 | 100.00 |
| Range | 1 mo to 12 yrs | | 1 mo to 10 yrs | | 1 mo to 12 yrs | |
| Median | 13.5 mo | | 12 mo | | 12 mo | |
| Mean | 44.9 mo | | 32.8 mo | | 39.6 mo | |
| SD | 46.9 | | 37.6 | | 43.1 | |

Nearly half 47.3% were infants. Children between 1-5yrs and 5-10 years were 21.1% in each category. > 10 yrs children accounted for 10.5% of shock cases.

The mean age of study population was 12 months. The median ages of male and female were 13.5 months and 12 months respectively.

The sex wise mean age of males and females were 49.9 46.9 months and 32.8 37.6 month respectively. The difference between the age groups was not significant statistically. The mean age of total study subject was 39.6 43.1 months.

II. Prevalence of pediatric shock:

During the study period 2035 Pediatric patients were treated as inpatients. Among them 1189 were males and 846 were females. 57 of these children were diagnosed to have shock which makes upto 2.8%.

Table:2**Sex wise distribution of Pediatric shock cases**

| Sex | Total children admitted in ward / PICU | Total children admitted with shock | Percentage | Prevalence per 1000/p |
|------------|---|---|-------------------|----------------------------------|
| Male | 1189 | 32 | 2.7 | 26.9/1000 |
| Female | 846 | 25 | 3.0 | 29.8/1000 |
| Total | 2035 | 57 | 2.8 | 28/1000 |

The above table explains the prevalence as 28 /1000 patients. In males it was 26.9/1000 and in females it was 29.8/1000. The difference between the two groups was not statistically significant.

Among the 57 children diagnosed to have shock the following were the pattern of **clinical findings** observed.

| Clinical finding | No: | % |
|---------------------------------|----------------|----------|
| Unstable airway / Bradypnea | 19 | 33.3 |
| Effortless tachypnea | 24 | 42.1 |
| Respiratory distress | 23 | 40.4 |
| Tachycardia | 42 | 73.7 |
| Relative / Absolute bradycardia | 15 | 26.3 |
| CRT Prolonged | 52 | 91.2 |
| Flash refill | 5 | 8.8 |
| Blood pressure low | 33 | 57.9 |
| Liver span increased | 24 | 42.1 |
| Altered Mental Status (A/V/P/U) | 57 | 100 |
| Urinary output (>1ml/kg/hr) | 31 (Out of 38) | 81.6 |

All children who had unstable airway or bradypnea, were having decompensated shock and except one among them all expired despite prompt airway management. Respiratory distress noticed in 23 (40.4%) of children and all of them had either cardiogenic, septic shock or a combination of both. Capillary refill time was prolonged in 52 (91.2%) of children and the remainder 5 (8.8%) had flash refill and managed as warm septic shock. Decompensated shock as evidenced by low blood

pressure was seen in 57.9% children. All of them had altered mental status. Urinary output was monitored in 38 children of which 31 (81.6%) had oliguria.

SEVERITY OF SHOCK:

Based on severity 2 types of shock were recognized – Compensated and Decompensated.

Table 3: Severity of shock – percentage distribution

| Sex | Compensated | | Decompensated | | Total | | Significance |
|------------|--------------------|----------|----------------------|----------|--------------|----------|---------------------|
| | No | % | No | % | No | % | |
| Male | 10 | 17.5 | 22 | 38.6 | 32 | 56.1 | p>0.05 |
| Female | 14 | 24.6 | 11 | 19.3 | 25 | 43.9 | p>0.05 |
| Total | 24 | 42.1 | 33 | 57.9 | 57 | 100.00 | |

Table – 3 shows the sex wise distribution of the severity of shock. Among the 57 cases, 10 males (17.5%) and 14 females (24.6%) were compensated 22 males (38.6%) and 11 (19.3%) females were decompensated. The difference was not statistically significant.

42.1% (n=24) had compensated shock and 57.9% (n=33) had decompensated shock at the time of presentation

Table 4: Age wise distribution of severity of shock

| Age | Compensated | | Decompensated | | Total | |
|-------------|--------------------|----------|----------------------|----------|--------------|----------|
| | No | % | No | % | No | % |
| <12 mo | 10 | 17.5 | 17 | 29.8 | 27 | 47.3 |
| 1yr – 5yr | 5 | 8.8 | 7 | 12.3 | 12 | 21.1 |
| 5yr – 10yrs | 5 | 8.8 | 7 | 12.3 | 12 | 21.1 |
| >10 yrs | 4 | 7.0 | 2 | 3.5 | 6 | 10.5 |
| Total | 24 | 42.1 | 33 | 57.9 | 57 | 100.0 |

Infants were affected more by decompensated shock 17 (29.8%) than the other age group. Decompensated shock was seen in 33 (57.9%) children which though was greater than in compensated 24 (42%), the difference was not statistically significant ($p>0.05$).

Infants made upto about 47.3% of case of shock and also had a severe degree of the disease at presentation.

Table 5: Percentage distribution of cases – Etiology wise.

| Etiology | <12mo | | 1 – 5yrs | | 5 – 10yrs | | >10yrs | | Total | |
|------------------------|-----------------|----------|-----------------|----------|------------------|----------|------------------|----------|--------------|----------|
| | No | % | No | % | No | % | No | % | No | % |
| Septic | 11 | 19.3 | 4 | 7.0 | 0 | 0.0 | 1 | 1.8 | 16 | 28.1 |
| Cardiogenic | 1 | 1.8 | 0 | 0.0 | 4 | 7.0 | 2 | 3.5 | 7 | 12.2 |
| Hypovolemic | 4 | 7.0 | 1 | 1.8 | 3 | 5.3 | 1 | 1.8 | 9 | 15.8 |
| Septic/ Cardiogenic | 7 | 12.3 | 2 | 3.5 | 1 | 1.8 | 0 | 0.0 | 10 | 17.5 |
| Distributive | 4 | 7.0 | 5 | 8.8 | 2 | 3.6 | 2 | 3.6 | 13 | 22.8 |
| Anaphylactic | 0 | 0.0 | 0 | 0.0 | 1 | 1.8 | 0 | 0.0 | 1 | 1.8 |
| Neurogenic | 0 | 0.0 | 0 | 0.0 | 1 | 1.8 | 0 | 0.0 | 1 | 1.8 |
| Total | 27 | 47.4 | 12 | 21.1 | 12 | 21.1 | 6 | 10.5 | 57 | 100 |

The etiological classification was done as

- Hypovolemic
- Septic
- Cardiogenic
- Distributive
- Anapylactic
- Neurogenic.

There was also noticed a combination of more than one type of shock in a single case. Most of these were septic shock with cardiogenic involvement.

Among the 57 cases studied septic (19.3%) was the major type among infants and 28.1% among the total group. This did not include the Septic / Cardiogenic type which accounted for 17.5% of cases. Hypovolemic was seen in 15.8% of cases and distributive in 22.8% of case. Cardiogenic shock was seen in 12.2%. One child (1.8%) had anaphylactic shock and another one (1.8%) had neurogenic shock due to omam water poisoning.

Results of the outcome of management:

Children with shock admitted to our PICU were managed as per PALS guidelines. Airway management, oxygen administration was done for all children. Some children received only crystalloid, others required crystalloids and Inotropes. Some others also required Catecholamine support. Children with specific etiology as scorpion sting, DKA, sepsis, Bee sting anaphylaxis, Dengue shock syndrome were managed specifically according to their etiology.

Table: 6 Percentage distribution of management modalities among compensated Decompensated groups.

| Intervention | Compensated | | Decompensated | | Total | |
|--|-------------|-------------|---------------|-------------|-----------|------------|
| | No | % | No | % | No | % |
| Crystalloids alone | 9 | 15.8 | 2 | 3.5 | 11 | 19.3 |
| Crystalloids +Inotropes | 15 | 26.4 | 15 | 26.4 | 30 | 52.7 |
| Crystalloids + Inotropes + Epinephrine | 0 | 0.0 | 8 | 14.0 | 8 | 14.0 |
| Crystalloids+ Epinephrine | 0 | 0.0 | 8 | 14.0 | 8 | 14.0 |
| Total | 24 | 42.1 | 33 | 57.9 | 57 | 100 |

Children who were managed only with crystalloids were 11 (19.3%) and (49.2%) 28 of children with shock required inotropic support in addition to crystalloids. 8 children (14%) required initiation of Adrenaline infusion after fluid resuscitation directly as they were in post resuscitative stabilization phase. 8 children (14%) went on to become catecholamine resistant requiring Epinephrine infusion.

Among 11 children who required only fluids only 2 children were from the decompensated category. One child was AGE with severe dehydration and required 80ml/kg of RL and another was also AGE with severe dehydration who required 60ml/kg of RL.

Table: 7 Requirement of Intubation and Bag and mask ventilation

| Etiology | Compensated | | Decompensated | | Total Intubation Required | |
|-------------------------|-------------|-----|---------------|------|---------------------------|-------|
| | No | % | No | % | No | % |
| Septic | 1 | 5.3 | 6 | 31.6 | 7 | 36.9 |
| Cardiogenic | - | - | 1 | 5.3 | 1 | 5.3 |
| Hypovolemic | - | - | - | - | - | - |
| Septic / cardiogenic | - | - | 8 | 42.1 | 8 | 42.1 |
| Distributive | - | - | 3 | 18.7 | 3 | 18.7 |
| Anaphylactic | - | - | - | - | - | - |
| Neurogenic | - | - | - | - | - | - |
| Total | 1 | 5.3 | 18 | 94.7 | 19 | 100.0 |

19 children with shock were intubated which worked up to 33.3% of total children with shock. Among the 19 intubated children 18(94.7%) were decompensated and 1(5.3%) child had compensated shock. Only one among the 19 children survived.

Renal function and liver function tests in children with shock :

Renal function tests were done only in 50 children and liver function tests were done only in 45 children during the study due to difficulty in obtaining blood sample due to severity of shock while presentation and shorter duration of stay in the hospital.

Of the 50 children whose renal function test was available, 23 have elevated values – 17 from the decompensated category and 6 from the compensated. The difference was statistically significant.

($t = 2.23$ d.f = 55 and $p < 0.05$)

| RFT & LFT | Compensated | | Decompensated | | Total | |
|----------------------|--------------------|----------|----------------------|----------|--------------|----------|
| | No | % | No | % | No | % |
| RFT | 6 | 24 | 17 | 51.5 | 23 | 40.4 |
| LFT | 5 | 20.8 | 11 | 33.3 | 16 | 28.1 |

Of the 45 children whose liver function test was available, 16 have elevated values -11 from the decompensated category and 5 from the compensated category. The difference was not statistically significant.

($t = 1.072$ d.t = 55 and $p > 0.05$)

Death:

| | Compensated | | Decompensated | | Total | |
|----------|-------------|------|---------------|------|-------|------|
| | No | % | No | % | No | % |
| Improved | 20 | 83.3 | 12 | 36.4 | 32 | 56% |
| Died | 4 | 16.7 | 21 | 63.6 | 25 | 43.8 |

Total number of deaths among 57 cases was 25 (43.8%) – 21 out of 33 decompensated shock cases died (63.6%) and 4 out of compensated shock cases died (16.7%). This difference was statistically significant, ($t=4.143$ d.f =55 and $p<0.001$).

Outcome based on etiological classification:

| Etiology | Improved | | Died | | E | d.f | Significans |
|------------------------|-----------|------------|-----------|------------|-------|-----|-------------|
| | No | % | No | % | | | |
| Septic | 8 | 25 | 8 | 320 | 0.5 | 55 | p>0.05 |
| Cardiogenic | 6 | 18.8 | 1 | 4.0 | 1.864 | 55 | p>0.05 |
| Hypovolemic | 8 | 25 | 1 | 4.0 | 2.442 | 55 | P<0.05 |
| Septic/ Cardiogenic | 1 | 3.1 | 9 | 36.0 | 3.264 | 55 | P<0.01 |
| Distributive | 7 | 21.9 | 6 | 24.0 | 1.463 | 55 | p>0.05 |
| Anaphylactic | 1 | 3.1 | 0 | 0.0 | 2.171 | 55 | p>0.05 |
| Neurogenic | 1 | 3.1 | 0 | 0.0 | 1.011 | 55 | p>0.05 |
| Total | 32 | 100 | 25 | 100 | | | |

Death and improvement following management of shock were the two variables measured in study. Among the septic shock category 8 improved and 8 died. Among cardiogenic shock 6 improved and 1 died. Both there were not statistically significant. Where as in hypovolemic shock 8 improved and 1 died and the difference was statistically significant in children who had both septic + cardiogenic shock only 1 survived and 9 died which was also significant statistically.

DISCUSSION

Studies analyzing the demographic profile and prevalence of shock in pediatric patients who present to a tertiary care hospital are very few in both western and Indian literature.

Most Western literature as well as **Frankel LR, Mathers LH; Shock – In Nelson Textbook of pediatrics 17th edition** publish that approximately 2% of all hospitalized children are diagnosed with shock.

Indian study at **Dayanand Medical College Hospital, Ludhiana, Punjab, by Daljit Singh et al in Indian Pediatrics / July 2006, 43:619-623** who studied 98 shock cases out of 2274 admitted patients give their percentage as 4.3%.

In this study conducted at Tirunelveli Medical College, 57 cases of shock were registered out of the 2035 pediatric cases admitted during the study period, which works up to 2.8%.

47.3% (n=27) of the total shock cases were infants while in the study by Daljit Singh et al infants made up to 39.8% of total cases (n=39).

Mean age of study population in this study was 3.3 ± 3.8 years while in the study compared it is 2.8 ± 3.4 years.

Sex wise distribution of shock patients did not show any significance though of those children admitted, 846 were females and 1189 were males and 3% and 2.7 % of them respectively were diagnosed

to have shock. Neither did the severity of shock – compensated or decompensate have any difference among the two sexes.

But infants were affected more by decompensated shock at time of presentation than any other age group in this study but Daljit Singh et al study showed no significance to relation between age and severity of shock.

Analysis of clinical features revealed the following. All 57 cases were assessed by rapid cardiopulmonary assessment at presentation and the data of clinical findings obtained is discussed below.

The most consistent finding noticed in the cases was altered level of sensorium at presentation. This was done using the A/V/P/U scale. All children (100 %) had impaired consciousness of varying degrees.

Next common finding was that of decreased urinary output noticed in 81.6% of children. Only 38 children with shock were catheterized for monitoring urine output out of which 31 had oliguria.

Capillary refill time was prolonged in 91.2 % (n=52) and flash refill noted in 8.8 % (n=5). All these 5 children were among the warm septic shock category at presentation.

Tachycardia surprisingly was seen in only 73.7% (n=42) children. The rest had relative / absolute bradycardia.

Respiratory problems ranged from bradypnea, respiratory arrest, effortless tachypnea to respiratory distress.

Respiratory distress was seen in 40.4% (n=23) children and all of them had septic / cardiogenic shock.

Unstable airway / bradypnea was noticed in 33.3% (n=19) and these children were having decompensated shock / imminent arrest.

57.9 % (n=33) cases of shock were decompensated while presentation to this hospital in while only 40% (n=39) cases were decompensated in the study conducted by Daljit Singh et al. In our study Children presented to the hospital in a more severe degree of shock.

63.6% (n=21) of the 33 compensated shock cases died and 16.7% (n=4) of the 24 compensated shock cases died in our study while the percentage of death among the two groups was 67% and 2% respectively in the Punjab Study by Daljith Singh et al.

Septic and cardiogenic shock accounted for 37.8% of total shock cases while septic shock alone accounted for the single most common form of shock among the cases in 28.1% (n=16).

Also Septic shock was the major form of shock among the infants accounting for 19.3% (n=11) the infants. Septic and cardiogenic causes were seen in upto 33.4%(n=19) of infants with shock.

The next common form of shock noticed was distributive shock which accounted for 22.8% (n=13) of 57 cases of shock. All these cases were suspected and later proved to be children with Dengue shock syndrome or Dengue hemorrhagic shock.

Hypovolemic shock came in next with 15.8% (n=9) of cases. All of them were due to diarrheal dehydration.

One case of anaphylactic shock due to multiple bee sting was admitted in decompensated shock and responded well to isotonic fluid replacement, IM adrenaline and IV Hydrocortisone.

One case of neurogenic shock was a result of Omam water poisoning and the child succumbed to decompensated shock.

In the study by **Chang et al 1999 Critical Care pediatrics 1999. Outcome of Pediatric Shock** – 22 cases, were studied of which 50% (n=11) was due septic shock as compared to 45.6% (n=26) in our study. 7 were due to hypovolemia and 4 were due to carcinogenic shock.

End points of management were achieved with isotonic fluids alone in 9 (15.8%) of cases with compensated shock and 2 (3.5%) of cases with decompensated shock. These two children who had received more than 80ml/kg of isotonic fluids were hospitalized with severe diarrheal dehydration.

Dopamine in addition to isotonic fluids was administered to achieve end points in 14 (24.6%) with compensated shock and 13 (22.8%) of patients with decompensated shock.

Adrenaline infusion was used in 16 children (28%) of which 8 were administered Adrenaline following post arrest stabilization and 8 were administered Adrenaline infusion because they were catecholamine resistant. All the 16 children were in decompensated group.

Intravenous Hydrocortisone was used in 5 children with septic decompensated shock who were resistant to inotropic support. Inodilators were not used in our study.

Intensive care medicine 32, 7.7.06, 995-1003 article titled. Fluid resuscitation in Hypovolemic shock has concluded there is a significant decrease in mortality when > 40ml /kg of fluids were administered in the first hour hospitalize.

In our study 40 children (70.2%) out of the 57 cases had received >40ml / kg of fluid resuscitation in the first hour of management of these 40 children 20 of them died of which 85% (n=17) and 15% (n=3) of them suffered from decompensated and compensated shock respectively. Remaining 20 of those children survived.

19 (33.3%) of 57 children required endotracheal intubation and one more child required bag and mask ventilation. All of these children 94.7% (n=18) were among the decompensated group except for one child

5.3% (n=1) who was compensated at time of presentation. Only one child of the 19 requiring intubation survived.

Liver function tests were elevated in 28.1% (16 out of 45) of children with shock and no significant difference was found between the compensated and decompensated groups.

Renal function tests were elevated in 40.4% (23 out of 50) of children with shock and a significant difference was noticed with more children from the decompensated category having increased values.

Death occurred in 43.9 % (n=25) of 57 cases of shock when compared to 26.4% (n=31) of 98 cases in the **Punjab study by Daljit singh et al** statistically significant improvement among etiological classification was seen with children in the Hypovolemic group and significant no of deaths occurred in the group which had features of both septic and cardiogenic shock.

CONCLUSION

Shock constitutes a significant percentage of diagnosis in critically ill children. Infants are affected by shock and have severe degree of shock at diagnosis than more than any other age group in the study. No difference in prevalence or severity of shock at presentation between the two sexes was noticed. Septic shock accounts for majority of decompensated shock and poor outcome to management. Infancy decompensated shock, septic shock and those requiring ventilatory support were the factors influencing the outcome of management.

BIBLIOGRAPHY

- 1) Ayse Aklan Ariban , Agob Citak – Pediatric shock – Signae vitae
2008 3(1) 13-23
- 2) Chang – Outcome of Pediatrics shock ,Critical Care Pediatrics 1999.
- 3) Carcillo et al – Fluid in septic shock – JAMA 1991 266 1242-45.
- 4) Fluid resuscitation in Hypovolemic shock – intensive care
Medicine 32 7.7.06 995 – 1003.
- 5) Pediatrics on call – Praveen Khilani
- 6) Pediatrics on call – Daljit Singh et al – 15.5.2006
- 7) Pediatric Emergency Medicine Course manual – Shock – 45- 62
2008
- 8) PALS provider manual 2002:30 -40 127- 146
- 9) Tobin JR.Wetzel RC. Shock and multiorgan system failure.
Textbookof Pediatric Intensive Care. 3 rd Edn, Roger MC.
- 10) Frankel LR .Mathers LH'Shock –In Textbook of Pediatrics Nelson
17 th edn.
- 11) McConnel MS Perkin RM . Shock states. In Pediatric Critical Care.
2nd edn. Fuhrman BP , Zimmerman JJ. St. Louis, Mosby 1998; 293
– 305.
- 12) Adam. J. Schwarz [emedicine.com/PED/topic 3047](http://emedicine.com/PED/topic/3047).

- 13) Tobin JR, Wetzel RC. Shock and multiorgan system failure. Textbook of Pediatric Intensive Care. 3rd Edn, Roger MC
- 14) American Heart Association. Recognition of shock and respiratory failure. In Chameides L, Hazinski MF. Pediatric Advanced Life Support.
- 15) Carcillo JA, Fields AI, American College of Critical Care Medicine Task Force Members. Clinical practice parameters for hemodynamic support of pediatric and neonatal patients in septic shock, Crit Care Med. Jun. 2002; 30(16) 1365 – 78
- 16) Bollaret FE, Baeur P, Audibert et al. Effects of epinephrine on hemodynamics and oxygen metabolism in dopamine resistant shock. Chest 1990 – 98 949-53
- 17) Notterman D. Inotropic agents, Crit Care Clin 1991 7: 583
- 18) Murphy K. Pediatric Triage Guidelines St. Louis Mosby 1997.
- 19) American Academy of Pediatrics. Pediatric Education for Prehospital Professionals. Elk Grove village IL Jones and Barlett 2000.
- 20) Praveen khilani, Pediatrics on call.
- 21) Archives Disease of childhood 2008; 85(5) 386 -90 Booy R Habibi P Nadel.
- 22) Fluid resuscitation in Hypovolemic shock, Intensive care medicine 32 7.7.06 995-1003.

- 23) Irwin and Rippe's intensive care medicine, Philadelphia, Lippincott Williams and Wilkins.
- 24) Fleisher G. Ludwig S: Textbook of Pediatric Emergency Medicine 4th ed Philadelphia Lippincott.
- 25) Cochrane Injuries Group Albumin Reviews BMJ 1998; 317: 235-240.
- 26) N Engl J med 2004;350:2247-2256: SAFE study investigator – A Comparison of albumin and saline for fluid resuscitation in intensive care unit.
- 27) Lancet 2003;362:1320-23 Duke T, Molyneux EM. Iv fluids for seriously ill children.
- 28) Ewarch Infect Diseases. 2005;6-6 Resuscitation in Dengue shock syndrome ; Crystalloids or Colloids equally effective.
- 29) Singhi S. Shock In Sachdev HPS. et al Principles of Pediatric and Neonatal emergencies 2nd edn. New Delhi: Jaypee. Medical Publishers.
- 30) Cotran RS, Kumar V, Robbins SL. Shock in Fluid and Hemodynamic Derangements. In: Robbins Pathologic Basis of Disease. WB Saunders 1989 114-119.
- 31) Carcillo JA, Kuch BA, Han YY, Day S et al in Pediatrics 2009 Aug; 124 (2) 500-8, e pub 2009 Jul 27. Department of Pediatrics and critical care Medicine, University of Pittsburgh School of Medicine.

32) Ped Crit Care Med 2003 Oct; 4(4): 471 – 5 Ringe – HI, Varnholt V
Gaedicke G PICV. Charite Children's hospital Humboldt V Berlin.

Proforma for dissertation in Pediatric Shock

S.No.:

Date:

Patient Name :

IP No:

Age :

DO Admn.

Sex :

Final diagnosis:

History :

| | | | |
|------------------------|--|----------------------|--|
| FEVER | | COUGH & COLD | |
| LETHARGY | | RESPIRATORY DISTRESS | |
| COOL EXTREMITIES | | CYANOSIS | |
| DECREASED URINE OUTPUT | | LOOSE STOOLS | |
| ALTERED SENSORIUM | | TRAUMA | |
| SEIZURES | | BLEEDING | |
| POSTURING | | ABSCESSSES | |
| DRUG INTAKE | | SCORPION STING | |
| BEE STING | | SNAKE BITE | |

Past history :

Known heart disease

Chronic /recurrent diarrhea

On Examination:

Airway: Stable/Unstable Maintainable or not

Respiratory rate:

Stridor Grunting SCR/ICR

Air entry Creps/wheeze Color

Heart rate

SIS2 Murmur Cool below

| | | |
|-------------------------------|------------|--------------|
| Central peri.pulse difference | | CRT |
| BP | Liver span | Urine output |
| GCS | Pupils | DEM/EOM |
| Tone | Seizures | Posturing |

Treatment details :

| | | |
|-----------------------------|---------|-------------|
| O2 | BVM | Intubations |
| IVF | ml over | |
| IV Dopamine/Dobutamine | | |
| IV Adrenaline/Noradrenaline | | |
| IV Vasodilators | | |

INVESTIGATIONS:

| | | | | |
|---------------|------|---------------|----------|---|
| Sugar | Urea | Creatinine | Na | K |
| Bilirubin | SGOT | SGPT | Proteins | |
| Blood culture | | Urine culture | | |
| CXR | | ECG | Other | |

Course of illness while management

| | | | | | | | |
|---------------------------|--|--|--|--|--|--|--|
| Time | | | | | | | |
| Airway | | | | | | | |
| RR | | | | | | | |
| Grunting | | | | | | | |
| SCR/ICR | | | | | | | |
| Air entry | | | | | | | |
| Creps/wheeze | | | | | | | |
| Color | | | | | | | |
| SaO2 | | | | | | | |
| HR | | | | | | | |
| Cool below | | | | | | | |
| Central peri Pulse diffn. | | | | | | | |

| | | | | | | | |
|---------------|--|--|--|--|--|--|--|
| CRT | | | | | | | |
| BP | | | | | | | |
| Liver span | | | | | | | |
| GCS | | | | | | | |
| Pupils | | | | | | | |
| DEM/EOM | | | | | | | |
| Tone | | | | | | | |
| Posturing | | | | | | | |
| Seizures | | | | | | | |
| Urine output | | | | | | | |
| Interventions | | | | | | | |

Outcome: Etiology :

Classification :

MODS :

Outcome of therapy :

Investigator:

Title: _____

IP No: _____

Telephone: _____

Date form completed: _____

